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Lipoprotein(a) Concentrations in Non-Selected Hospitalised Patients Between 18 and 100 Years of Age: Comparison with Cholesterol Fractions and Triacylglycerols in Patients with Lipid Status Requests

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Summary: In a study designed to measure lipoprotein(a), cholesterol, cholesterol fractions and triacylglycerols in serum 4004 hospitalised individuals aged between 18 and 100 years were examined.

Lipoprotein(a) was determined in 1313 patients (438 males, 875 females) aged 18–59 years and 489 patients (234 males, 255 females) aged 60–100 years. Cholesterol, cholesterol fractions and triacylglycerols were determined in a further 2037 patients (1084 males, 953 females) aged 18 to 100 years, for whom a lipid-status request had been made.

Lipoprotein(a) concentrations in 619 females measured directly postpartum were not significantly different from aged-matched female in-patients (n = 104) and age-matched female hospital staff (n = 114). Lipoprotein(a) concentrations in women (n = 77) aged 30–74 undergoing chronic haemodialysis were significantly higher ($p < 0.001$) than in men (n = 95) of the corresponding age group.

Median lipoprotein(a) serum concentrations showed a peak between 60–69 years in both men and women, i. e. at times of reported increased cardiovascular disease in both sexes. The lipoprotein(a) levels found in old age are comparable with those found in children and adolescents.

The lipoprotein(a) patient group was assessed according to age and clinic. Eight groups of patients were analysed. The maternity patients were significantly younger (median age 26 a) than the other seven groups ($p < 0.05 - < 0.01$), the hospital employees (median age 31 a) attending the annual check-up being younger than the remaining six groups ($p < 0.01$).

Lipoprotein(a) concentrations were marginally higher ($p = 0.05$) in the dialysis patients, when compared with those on internal medical wards.

Of the 'traditional' lipid analytes, the ratio LDL-cholesterol : HDL-cholesterol was of interest, being significantly higher in males aged 70–79 years of age, when compared with males under 30 years of age. Triacylglycerols were higher in men aged between 30 and 59 years ($p = 0.05 - < 0.01$).

The relationship between median analyte concentration and age was different for lipoprotein(a) than for the ratio LDL-cholesterol : HDL-cholesterol and triacylglycerols, thus further supporting the fact that lipoprotein(a) may be an independent risk factor for the development of atherosclerotic disease.

Introduction

The present trend toward establishment of reference intervals for hospitalised patients, although controversial (1, 2), emphasises the need for reliable data both from healthy and sick individuals, especially for laboratory analytes, in the management and diagnosis of disease.

The present article presents data from a population of patients aged between 18 and 100 years who were admitted to hospital for a variety of reasons. Specific groups studied included patients undergoing chronic haemodialysis, women directly postpartural, as well as patients from medical, surgical, urological and psychological wards and hospital employees attending an annual check-up. Lipoprotein(a) concentrations were measured in serum or plasma. In addition, cholesterol, LDL-cholesterol, HDL-cholesterol and triacylglycerols, were measured in another group of patients who had lipid-requests, with the aim of establishing age- and sex-dependent concentration ranges for both groups.

The German Federal Statistics Bureau (3) has reported a net increase in serum cholesterol levels in elderly patients, concomitant with a reduction in the mortality rates in the ICD 9 categories 410–414, i. e. deaths due to cardiac disease. As cholesterol is linked with atherosclerotic disease, it was of interest to study the concentrations of this analyte, (as total cholesterol, high- and low-density lipoprotein cholesterol and the ratio between low density and high density lipoprotein cholesterol), as well as lipoprotein(a), as both are ascribed major roles in the development of cardiovascular disease.

Materials and Methods

Patients

A total of 4004 persons was included in this study which was carried out between July 1993 and May 1995. The distribution of those studied was: 1313 patients between 18 and 59 years of age (438 males, 875 females) and 489 patients (234 males, 255 females) of age between 60 and 100 years (median age: males – 72 a [range 60–100 a], females – 75 a [range 60–93 a]) who underwent hospital treatment were admitted to the study for lipoprotein(a) determinations. Healthy hospital employees (n = 165) attending a routine check-up (51 males (median age 32 a) and 114 females (median age 31 a), 619 mothers directly after the birth of a child (median age 26 a) and 214 dialysis patients before dialysis (114 males, median age 53 a, 100 females, median age 62 a) were examined as separate groups.

In addition, the age of the patients and their lipoprotein(a) concentrations were analysed according to the clinic in which the treatment was being given. Eight groups were formed: medical, surgical, urological, psychiatric, maternity, haemodialysis, varia (clinics with too few patients for statistical analysis) and hospital staff attending an annual check-up.

The measurement of cholesterol, cholesterol fractions and triacylglycerols was performed in 693 patients with lipid-electrophoresis requests (378 males [median age 51 a] and 315 females [median age 58 a]) and 1344 lipid requests (247 males under 60 a [median age 51 a], 171 females under 60 a [median age 48 a], 459 males over 60 a [median age 66 a] and 467 females over 60 a [median age 68 a]). These groups were analysed separately from the lipoprotein(a) patients.

Assays used

Lipoprotein(a) was determined by means of immunoluminometric assay using two polyclonal antibodies directed against apolipoprotein(a) as already published in this journal (4, 5). One lot of reagents was used throughout the study, so that consistency of results is guaranteed.

Cholesterol was measured using the cholesterol oxidase-peroxidase-phenol-4-aminoantipyrine (CHOD-PAP) method (Boehringer Mannheim, Mannheim, Germany), which makes use of the *Trinder* reaction (6) to generate a colour in the visible spectrum.

HDL-cholesterol was analysed enzymatically as above after treatment of the serum with freshly prepared magnesium chloride-phosphotungstic acid reagent, (quantitative precipitation of chylomicrons, VLDL-cholesterol, LDL-cholesterol and lipoprotein(a)) and subsequent determination of cholesterol in the supernatant after centrifugation.

LDL-cholesterol was measured enzymatically as above after precipitation with dextran sulphate, which was supplied ready to use from Immuno, Heidelberg, Germany. The supernatant containing the HDL-cholesterol and VLDL-cholesterol was measured, the LDL-cholesterol concentration being determined from the difference (total cholesterol – supernatant cholesterol).

Triacylglycerols were analysed with a fully automated enzymatic method consisting of cleavage of the ester bonds and determination of the free glycerol by colorimetric detection using glycerol-3-phosphate oxidase, 4-chlorophenol, 4-aminophenazone and peroxidase (Boehringer-Mannheim – GPO-PAP method).

Quantitative lipid electrophoresis was performed using the Rapidophor electrophoresis system from Immuno, (Lipidophor all in 12). After electrophoretic separation, the lipoproteins were precipitated using a polyanionic solution followed by densitometric analysis of the electropherogram.

Statistics

As the distribution of lipoprotein(a) values was highly skewed (*Kolmogorov-Smirnoff* test), non-parametric statistics were used throughout. The median was used as central tendency and relevant percentiles as dispersion markers. The *Mann-Whitney* U-test was used for the comparision of two independent variables, the *Kruskal-Wallis* one way anlaysis of variance for the 14 groups followed by the *Nemenyi* test to detect differences between any two groups.

The data for the lipid analytes were checked for each analyte. The *Kolmogorov-Smirnoff* test showed that the analyte groups taken as a whole, irrespective of age and sex, were not normally distributed. The number of data in each age group was, however, often too small for a meaningful analysis of distribution, so that non-parametric statistics were performed, as for lipoprotein(a).

To compare the age groups of the patients in different clinics, parametric statistics were used as the data were normally distributed. A one way analysis of variance was performed to detect differences between the eight patient groups, followed by a modified least squares difference analysis as described by *Bonferroni*. The median and relevant percentiles were again used as markers of central tendency and dispersion.

Box and whisker plots were used to display the data visually. The interquartile range, 95% confidence limits, outliers (values lying between 1.5 and 3 box-lengths above the 75th percentile) and extreme values (more than 3 box-lengths above the 75th percentile) were shown for each box and whisker plot.

Results

Distribution of lipoprotein(a) concentrations

The use of p-values for the inter-group comparisons has been reserved only for statistically significant differences at the 5% and 1% levels.

Figure 1 shows the distribution of median lipoprotein(a) concentrations according to age and sex for the 18–100 year old persons. Table 1 shows the comparison between mothers at birth and non-pregnant hospitalised women as well as the renal insufficiency patients before haemodialysis. Table 1 also shows the quartile and median values, as well as the range of concentrations for each age group.

The median concentrations for males decline from a maximum in persons aged between 60 and 69 years to values similar to those in young children (4, 7) in those above 80 years of age. The difference between the 60–69 and 70–79 year old men was not statistically significant, but it was significantly lower in the 80–89 year old group ($p < 0.05$), compared with the 60–69 year old group. The pattern in women is different, with the highest values between 60 and 70 years, a decrease over the next decade, a small, but not statistically significant, increase between 80 and 90 years, and sharp decrease in women above 90 years of age, although the latter group was small ($n = 5$). In contrast to the men, the difference between the 60–69 year old women was not significantly different from the 80–89 year old group. There was a marginal significant increase in women under 30 years of age compared with women aged between 60 and 69 years ($p = 0.05$).

In the 18–59 year old group, the following statistical data were established. In the 18–29 year old women there was no difference between lipoprotein(a) concentrations in healthy non-pregnant women and either women at term, or women undergoing hospital treatment.

The same was true for the groups of women aged 30–39 years with the comparison between healthy non-pregnant women and women at term and those undergoing hospital treatment (tab. 1).

As a result of these analyses, all data for women between 18 and 59 years of age were assessed according to age, and independent of diagnosis.

There was no statistically significant difference in lipoprotein(a) concentrations between healthy and hospitalised males between 18 and 39 years of age so that no differentiation between healthy individuals and those undergoing hospital treatment was made. The data were treated just as for the females, i. e. in groups according to age only. The number of healthy men between 40 and 59 years was too small for a meaningful statistical analysis.

There was an increase in lipoprotein(a) concentrations in the 30–39 year old men compared with the 18–29 year old men, which was, however, not statistically significant ($p > 0.05$). A significant difference was seen when the 18–29 year old men were compared with 60–69 year old men ($p < 0.05$), but there was no significant difference when compared with the 40–49 and 50–59 year old men. There was no significant difference between the 30–39 year old men and the 40–49 year old men.

There was no difference in lipoprotein (a) concentrations in women between 18 and 29 and those between 30 and 39 years of age. There was a significant difference, however, between the 18 and 29 year old women and the 60–69 year old women $p = 0.05$) but not between the 18 and 29 old women and the 50–59 year old women.

There was no significant difference in lipoprotein(a) concentrations between men and women aged 18–79 years. The levels in men between 80 and 90 years of age were significantly lower than in women ($p = 0.01$). The number of subjects above 90 years of age was too small for statistical analysis (2 men and 5 women).

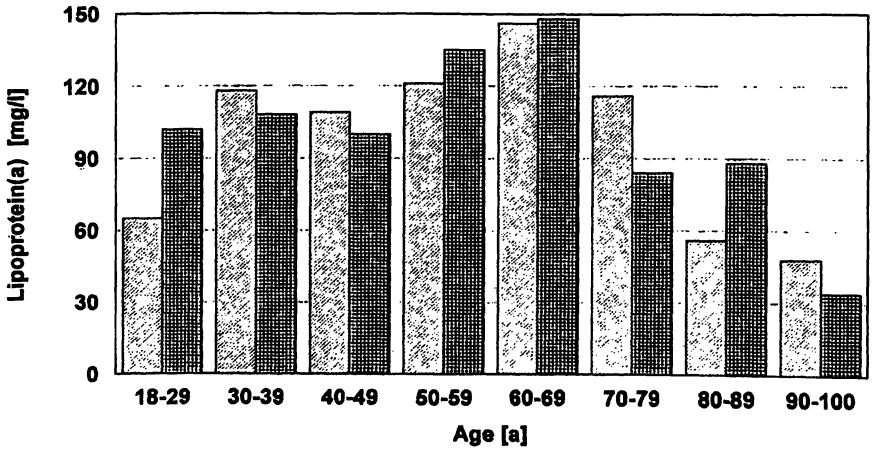

Fig. 1 Distribution of median lipoprotein(a) concentrations in the serum from 1967 adults aged between 18 and 100 years. The age groups are shown on the abscissa, the median concentrations for males ▨, and females ▩, on the ordinate.

The differences in lipoprotein(a) concentrations in the dialysis patients were unexpected (tab. 1). In women between 30 and 74 years of age undergoing dialysis, the serum lipoprotein(a) concentrations were significantly higher than in men of the corresponding age group ($p < 0.01$ in the age groups 30–44, 45–59 and 60–74 a).

Figure 2 shows the distribution of values in the 14 age groups studied. Due to the large spread of results it is small wonder that the differences in the median concentrations shown in figure 1 are rarely statistically significant.

Distribution of age groups and lipoprotein(a) concentrations according to clinics

Figure 3a shows the distribution of the ages in the different groups according to the clinic in which the patients were undergoing treatment.

The women on the maternity ward were significantly younger (median age 26 a) ($p < 0.05 - < 0.01$) than in the other seven groups. The hospital staff attending the annual check-up were younger than all groups except those on the maternity ward (median age 31 a) ($p < 0.01$). Table 2 shows the median ages of each

Tab. 1 Quartile values for lipoprotein(a) concentrations in the different groups studied.

	Lipoprotein(a) (mg/l)					
	Lowest value	1st Quartile	Median	3rd Quartile	Highest value	n
Males						
<30 years	5.9	33	54	150	1000	80
30–39	2.6	35	118	424	2576	146
40–49	2.7	35	109	368	1420	125
50–59	3.5	37	121	262	1069	87
60–69	2.0	43	146	359	2800	128
70–79	1.8	63	116	374	1907	65
80–89	4.0	39	56	102	521	41
>90	31		48		64	2
Females						
<30 years	1.0	41	102	286	3149	517
30–39	3.4	36	108	388	1630	224
40–49	1.0	56	100	494	2589	51
50–59	6.5	77	135	568	1976	83
60–69	2.2	38	148	370	1134	82
70–79	2.8	46	84	253	1345	87
80–89	4.3	40	88	211	1000	86
>90	9.0		34		68	5
Maternity patients						
<30	1.0	40	99	271	3149	347
30–39	3.4	44	109	307	1630	162
Female patients						
<30	9.0	30	94	244	1455	54
30–39	5.0	23	108	445	977	50
Healthy females						
<30	8.0	42	96	242	1256	60
30–39	5.7	41	108	285	1614	54
Males-dialysis						
<30	21		80		502	6
30–44	8.0	36	116	318	1099	27
45–59	3.5	57	140	320	1002	36
60–74	1.8	20	123	319	1907	32
75–89	14	64	89	289	880	13
Females-dialysis						
<30	3.2		107		535	3
30–44	17	54	250	502	2589	13
45–59	18	92	222	577	1976	28
60–74	2.8	142	290	511	1226	36
75–89	11	31	60	138	1164	19

group, together with the relationship between the mean and the median ages.

The average age of the patients attending the internal medical and urological clinics was higher than in the other groups.

Figure 3b shows the lipoprotein(a) concentrations in the same groups. The lipoprotein(a) concentrations in the haemodialysis patients (median 183 mg/l) were marginally higher ($p = 0.05$) than in those patients on the internal medical wards (median 100 mg/l). Again, the wide range of results reflects the minimal statistically significant difference between the groups. Table 2 shows the median lipoprotein(a) concentration for each group together with the value mean/median as an index of skewness of the data.

The difference in the number of subjects in figure 2 (n = 1802) and in figures 3a and 3b (n = 1756) is due to out-patients who could not be assigned to a definite clinic.

Distribution of cholesterol and triacylglycerols

A direct comparison of these groups of patients with those above is not statistically valid, as other patients were examined, who had had a serum lipid request, either directly in serum or as a lipid electrophoresis. Table 3 summarises the data shown below. Emphasis was laid on the ratio between low density- and high density lipoprotein cholesterol and on triacylglycerol levels in the three groups of patients studied.

These patients were first analysed as a single group, with the division between those under 60 and those over 60 years of age, according to sex, then in appropriate cases according to whether they had normal or abnormal lipid electrophoresis. Significant differences were found at the $p < 0.05$ level in the following cases. Total cholesterol (n = 1898) (fig. 4a) was higher in females between 30 and 39 and between 50 and 79 years of age, when compared with females under 30, and higher in males aged between 30 and 69 when compared with males between 70 and 89 years of age.

Triacylglycerols (n = 1966) (fig. 4b) were higher in males between 40–59 than in males aged 70–89, and higher in females aged between 60 and 69 years compared with females under 30.

HDL-cholesterol (n = 1655) (fig. 4c) was higher in men aged under 30 and between 40–59 compared with men aged between 70 and 89 years 60–69. In women, HDL-

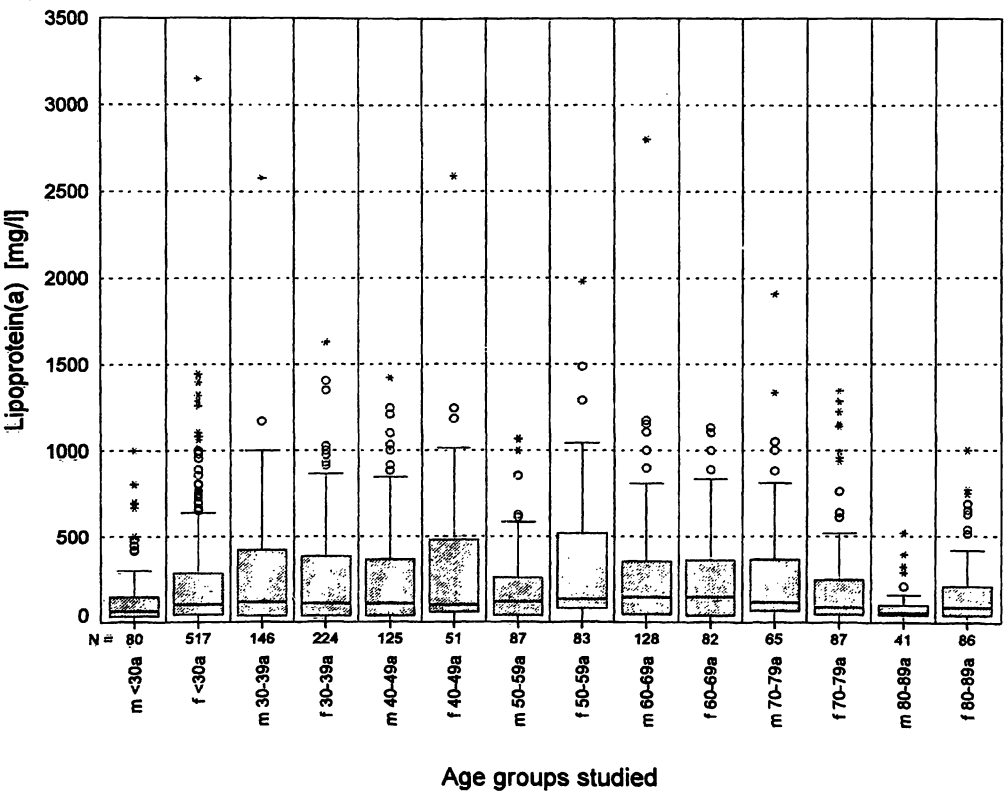

Fig. 2 The distribution of the lipoprotein(a) values of the 7 male (m) and female (f) age groups studied are shown using the box and whisker plot method.
The box and whisker plots show the data as follows: the box contains the results within the interquartile range, the bar within the box, the median valuc. The whiskers enclose the 95% confidence range. Outliers (between 1.5 and 3 box lengths above (or below) the box-limits) are shown as open circles, extreme values (values lying more than three box lengths from the box-limits) as asterisks. N denotes the number in each group.

cholesterol was higher in women aged 50 to 59 than in women aged 60–89 years.

LDL-cholesterol (n = 1646) (fig. 4d) was higher in men aged 50–59 compared with men aged 80–89 years. In

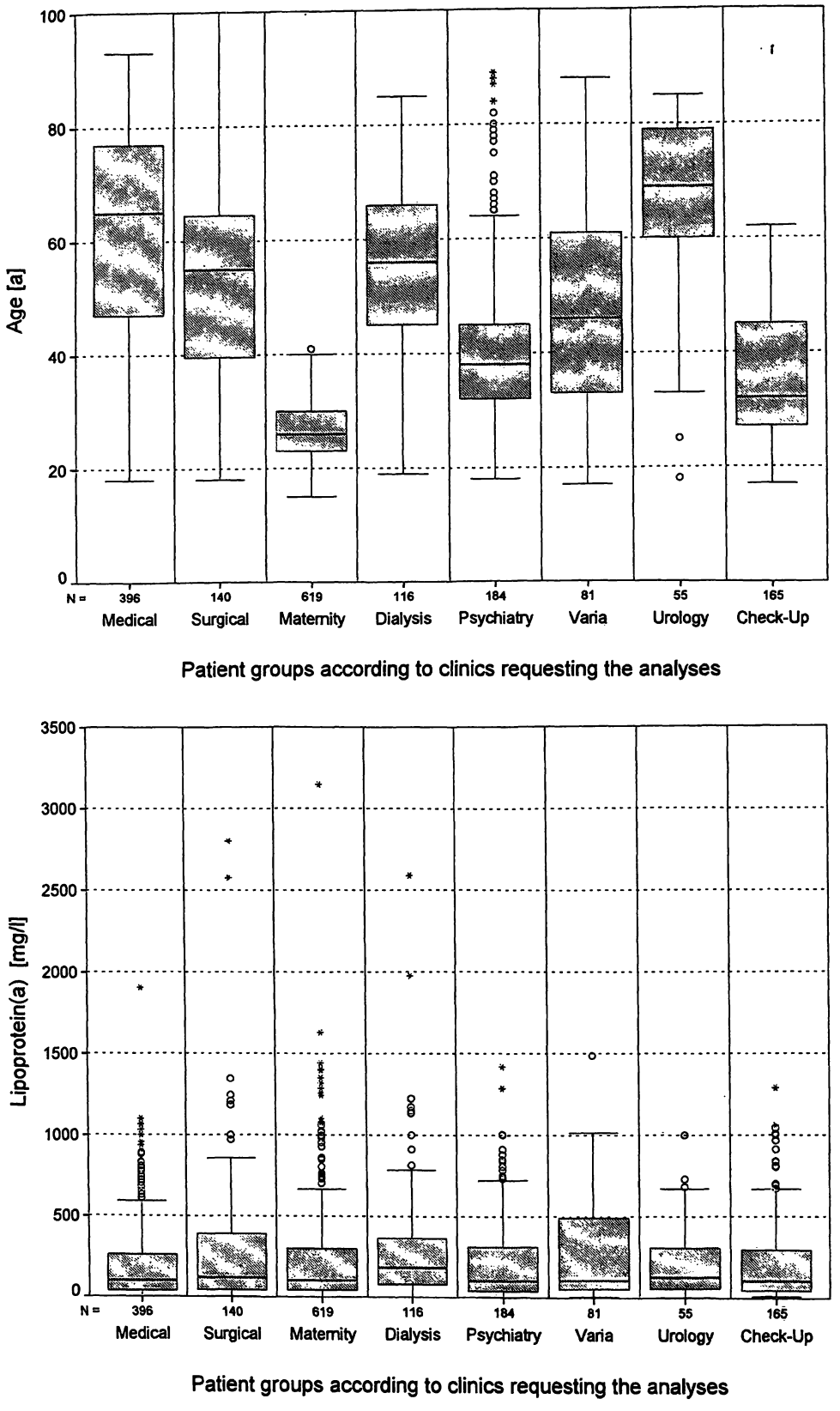

Fig. 3 a) Box and whisker plots
a) for the distribution of the age groups of the patients in the lipoprotein(a) group, according to the clinic in which they were being treated, the clinics being listed on the abscissa.
b) for the distribution of lipoprotein(a) concentrations.
The box and whisker plots show the data as follows: the box contains the results within the interquartile range, the bar within the box, the median value. The whiskers enclose the 95% confidence range. Outliers (between 1.5 and 3 box lengths above (or below) the box-limits) are shown as open circles, extreme values (values lying more than three box lengths from the box-limits) as asterisks. N denotes the number in each group.

Tab. 2 Frequency distribution of the patients according to the clinics requesting the analysis. In addition, age and median lipoprotein(a) (Lp(a)) values are given.

a) Lipoprotein(a) study

Clinic	Frequency (% of all patients)	Median age (years)	Median Lp(a) (mg/l)
Internal medicine	22.6	66	100
Surgical	8.0	55	121
Maternity	35.2	26	103
Nephrology/Dialysis	6.6	56	183
Psychiatry	10.5	38	103
Varia	4.6	46	107
Urology	3.1	69	128
Check-up	9.4	32	100

b) Lipid analytes study

Clinic	Frequency (% of all patients)	Median age (years)
Internal medicine	38.3	58
Nephrology/Dialysis	49.9	54
Varia	6.5	31
Outpatients	5.2	48

women, higher levels of LDL-cholesterol were observed in women aged 50–69 years compared with women aged under 30 and between 80 and 89 years of age.

The ratio LDL/HDL-cholesterol (n = 1686) (fig. 4e) showed no significant differences.

The numbers in parentheses for each analyte show the number of data used in the calculations.

In the patients with normal lipid electrophoresis patterns (tab. 3) the number of patients in each age group was too small for separate statistical analysis. The only point to note was that the women were on average (median age) over 10 years older than the men. In the men with pathological lipid electrophoresis patterns (tab. 3), the ratio LDL/HDL-cholesterol followed a similar pattern to that for lipoprotein(a), but with peaks occurring a decade earlier. Cholesterol levels showed a different pattern, without pronounced peaks. In the women with a pathological lipid electrophoresis (tab. 3), the ratio LDL/HDL-cholesterol followed a similar, even if less pronounced pattern as in the men. As in the men, the cholesterol levels showed little or no correlation with age, but were on average lower in women. Triacylglycerols were higher in men under 70 than in women, and lower in men than in women in those over 70.

As was to be expected, the patients with pathological electrophoresis patterns had significantly higher concentrations of all lipid analytes except for HDL-cholesterol, where there was no significant difference between any of the groups.

The separate groups are shown in table 3 for completion of data presentation, although only those data in figures 4a–4e, i. e. from the combined groups, are used for visual presentation.

Age and clinic attendance in the lipid groups

Tables 2 and 3 show the distribution of the ages of those included in the lipid patient groups. Whereas the selection of the patients for the lipid study group was taken from the requests for lipid-analysis, those from the lipoprotein(a) group were selected to give more or less equal numbers in each patient group, and were selected independently of the primary diagnosis. This gave rise to the different proportions of samples from each clinic. The majority of samples in the lipid group came from the internal medical wards (38.3%) and from the nephrology and dialysis clinics (49.9%). The number of out-patients (5.3%) and all other wards together (6.5%) account for the remaining patients. The median age of the lipid-patients was not different for the nephrology/dialysis patients, and was slightly lower for the internal medicine patients (tab. 2).

Discussion

Although the persons in both study groups were not identical, the number of observations made allows for a statistical evaluation of analytes in hospitalised patients for the analytes and persons tested. The German Federal Bureau of Statistics confirms early that around half the deaths registered are due to cardiovascular disease and atherosclerosis (3). Several studies have associated serum cholesterol levels with risk of cardiac disease (8–12) and national studies have been and are being carried out in industrialised society to educate the population of the dangers of high serum cholesterol (9, 12–14) and with the aim of reducing serum lipids.

The role of lipoprotein(a) as an independent risk factor in atherogenesis is generally accepted (15), but because it is at present not possible to reduce the concentrations of this analyte in blood over extended periods of time, little has been undertaken except heparin extracorporal LDL precipitation (HELP) apheresis, which has to be carried out regularly to have any significant effect (16). Short and medium term effects on reducing serum lipoprotein(a) have been reported using niacin (17), tamoxifen (18) and plant extracts (19) and in non-treated hyperthyroidism (20). Hydroxymethylglutaryl-CoA reductase inhibitors (17) and fibrous diets (21), although efficient in reducing LDL-cholesterol levels, do not

significantly alter lipoprotein(a) concentrations, again highlighting the different metabolic pathway of the latter.

The difference in behaviour of lipoprotein(a), cholesterol fractions and triacylglycerols in the different age groups support the claim that lipoprotein(a) is a risk factor which is independent of that of total cholesterol, and LDL-cholesterol. The median levels of lipoprotein(a) during life reflect the major occurrence of cardiac disease, with a small peak in men between 30 and 40, and a maximum between 60 and 70 years of age, and in women a relatively broad peak after the menopause, i. e.

Tab. 3 Median values of "classical" lipid analytes in men and women with lipid-requests.

	Cholesterol (mmol/l)	Triacylglycerols (mmol/l)	HDL-cholesterol (mmol/l)	LDL-cholesterol (mmol/l)	LDL-cholesterol / HDL-cholesterol	n
Males						
<30 years	6.20	1.90	1.40	3.90	3.03	56
30–39	6.50	2.70	1.10	4.32	4.57	92
40–49	6.95	2.40	1.20	4.80	3.60	140
50–59	6.90	2.50	1.10	4.70	4.35	230
60–69	6.60	2.10	1.00	4.70	4.45	373
70–79	6.20	2.00	0.90	4.40	4.98	153
80–89	5.50	1.60	0.90	3.42	3.78	36
>90	5.20	2.25	1.00	3.20	3.20	2
Females						
<30 years	5.90	1.20	1.20	4.05	3.37	51
30–39	6.60	2.00	1.30	4.60	3.76	50
40–49	6.60	2.20	1.32	4.50	3.55	62
50–59	7.20	2.10	1.38	5.10	3.87	187
60–69	7.00	2.20	1.10	5.00	4.50	315
70–79	6.70	2.30	1.10	4.70	4.08	181
80–89	6.30	1.85	1.00	3.80	4.05	102
>90	6.60	2.90	1.10	4.70	4.27	4
Males – Normal electrophoretic pattern						
<30 years	4.90	1.80	1.50	3.30	2.29	5
30–39	4.45	1.35	1.05	3.25	2.60	10
40–49	4.25	1.05	1.40	2.65	1.98	12
50–59	4.90	1.80	1.30	3.50	2.29	19
60–69	5.50	1.60	1.30	3.80	2.92	9
70–79	5.20	2.70	1.35	3.50	2.70	2
80–89	4.70	2.00	1.30	3.00	2.31	2
Females – Normal electrophoretic pattern						
<30 years	4.20	0.90	1.20	2.90	2.33	8
30–39	5.95	2.00	2.05	3.65	1.80	2
40–49	5.55	1.30	1.90	3.25	1.76	2
50–59	5.25	1.75	1.45	3.55	2.44	8
60–69	4.65	1.35	1.10	3.30	3.00	10
70–79	4.35	2.30	1.20	3.00	3.08	6
80–89	5.10	1.30	1.20	3.50	2.91	6
Males – Pathological electrophoretic pattern						
<30 years	7.65	3.30	1.05	4.40	4.30	21
30–39	8.30	3.65	1.00	6.45	5.81	47
40–49	7.80	3.75	1.05	5.55	4.00	74
50–59	8.00	3.10	1.15	5.90	5.06	85
60–69	7.50	3.35	1.10	5.30	5.09	72
70–79	6.70	2.50	1.00	4.70	3.82	17
80–89	7.00	1.50	1.60	5.10	3.19	2
Females – Pathological electrophoretic pattern						
<30 years	6.20	1.20	1.00	4.70	3.97	13
30–39	7.10	2.20	1.25	4.80	4.29	28
40–49	7.55	2.35	1.30	5.60	3.70	22
50–59	7.50	2.30	1.40	5.50	4.40	95
60–69	7.80	2.80	1.30	5.80	4.46	67
70–79	7.55	2.90	1.35	5.30	3.93	38
80–89	7.00	3.00	0.95	4.95	5.51	9

between 50 and 70 years of age. A second small peak in women between 80 and 90 years of age has been observed, although the significance of this must be further investigated and may very well be regional in nature. One thing is certain from this study, and that is, those who survive to very old age have median lipoprotein(a) levels similar to those found in children and adolescents (4, 7). This again supports the fact that low levels of lipoprotein(a) are of no significant additional risk for myocardial infarction.

The only indicator from the 'traditional' lipid metabolites which reflects the risk of cardiovascular disease in

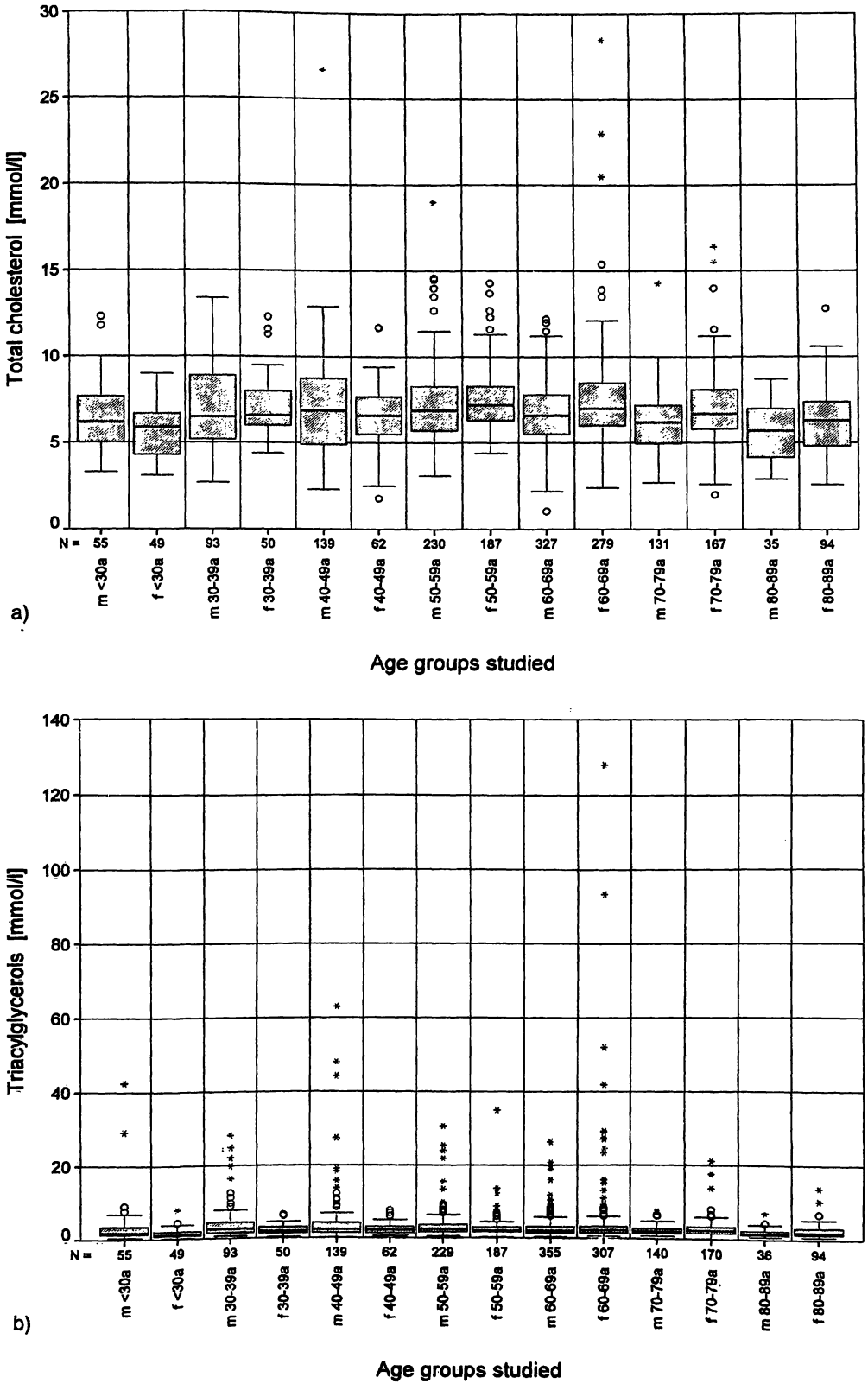

Fig. 4 Box and whisker plots for the lipid analysis observed in the 2037 patients with lipid requests.
a) total cholesterol
b) triacylglycerols

the patients with suspected or confirmed lipid disorders was the ratio LDL/HDL-cholesterol, although no statistically significant age-related differences were found with this quotient. The usefulness of this ratio is perhaps to be expected, as the negative (atherogenic) effects of LDL-cholesterol are further attenuated by a reduction in the partly atheroprotective effects of HDL-cholesterol. The use of LDL/HDL_2 or LDL/HDL_3 ratios appears to be too early, as the results on the atheroprotective effects of HDL_2 and HDL_3 are controversial (22).

The fact that the pattern for lipoprotein(a) compared with age, irrespective of the state of the individual (in

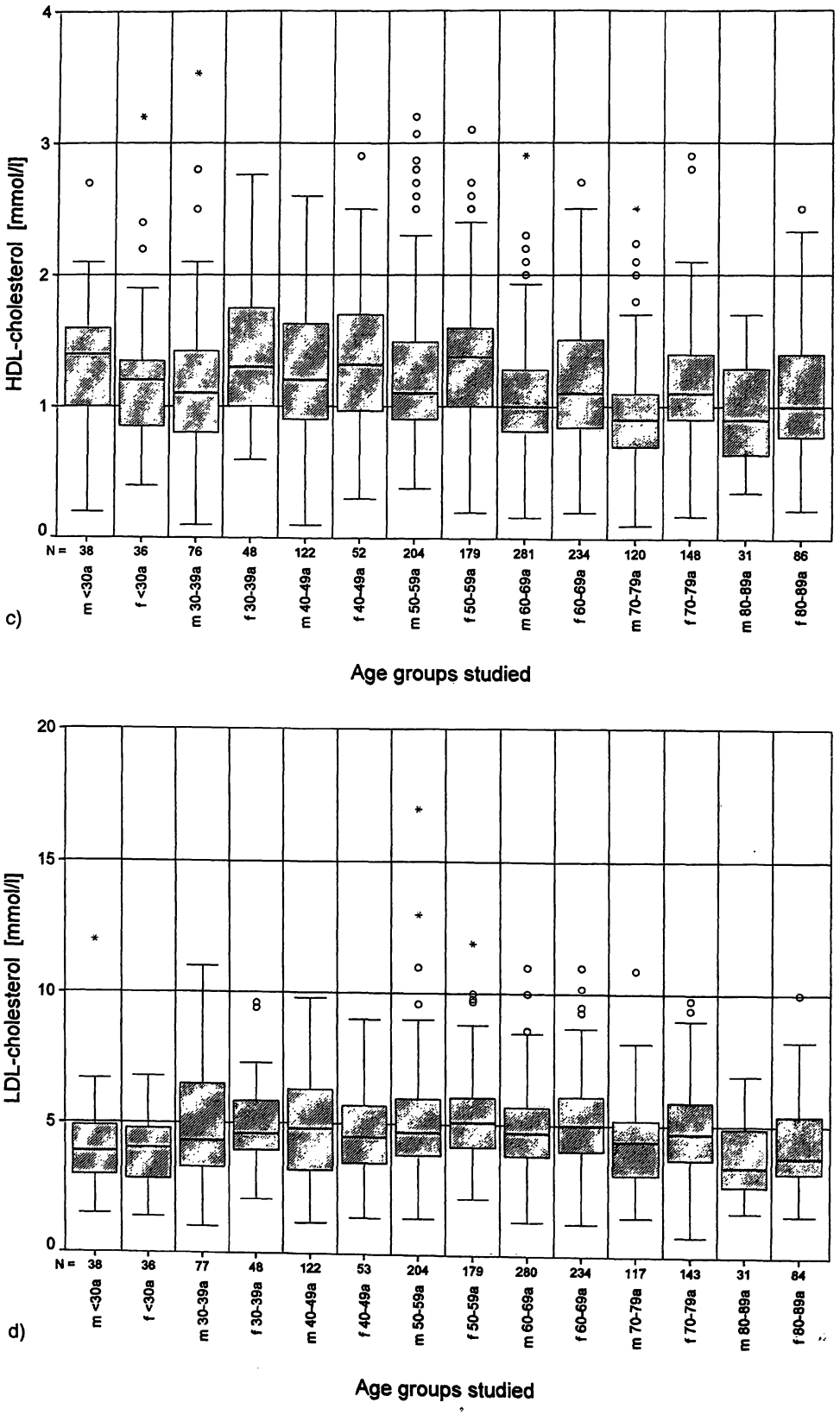

Fig. 4 Box and whisker plots for the lipid analysis observed in the 2037 patients with lipid requests.
c) HDL-cholesterol
d) LDL-cholesterol

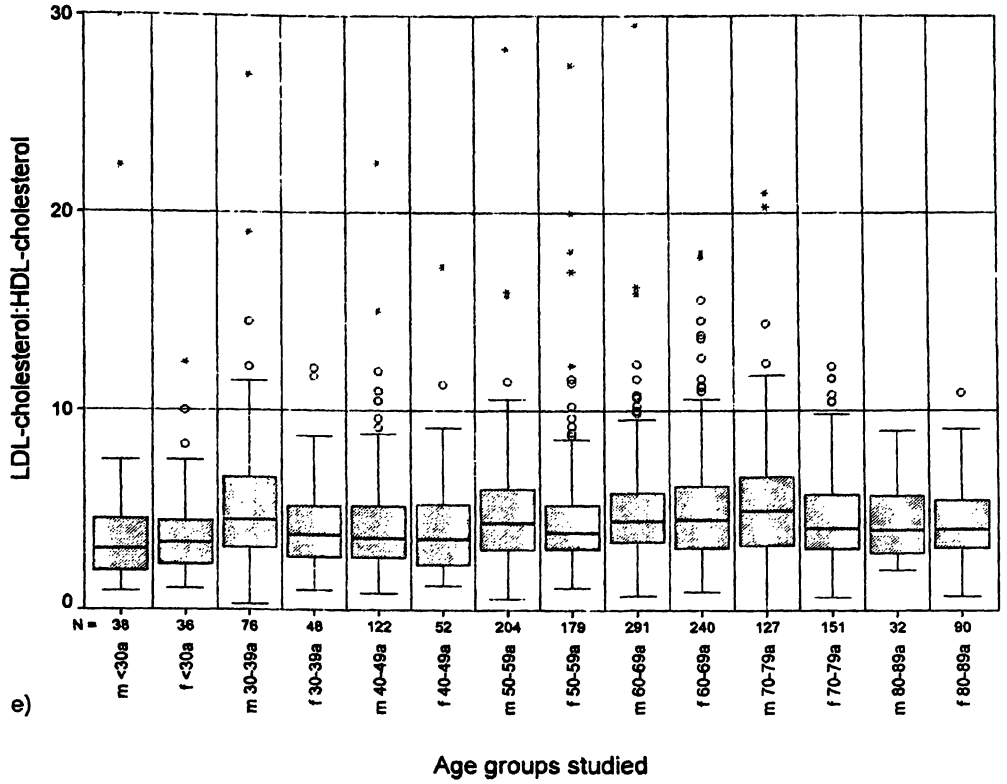

Fig. 4 Box and whisker plots for the lipid analysis observed in the 2037 patients with lipid requests.
e) the ratio LDL/HDL-cholesterol.
The data for figure 4e was from each individual LDL/HDL ratio, so that the median value shown may not be identical to that obtained by dividing median LDL-cholesterol by median HDL-cholesterol values.
The box and whisker plots show the data as follows: the box contains the results within the interquartile range, the bar within the box, the median value. The whiskers enclose the 95% confidence range. Outliers (between 1.5 and 3 box lengths above (or below) the box-limits) are shown as open circles, extreme values (values lying more than three box lengths from the box-limits) as asterisks. N denotes the number in each group.

this study no difference between mothers at birth, hospitalised women of the same age group and healthy non-pregnant controls) gives support to the genetic disposition rather than to acquired risk factors, as is the case for cholesterol-fractions in individuals not suffering from a genetic defect in cholesterol metabolism.

The findings that female haemodialysis patients above 30 years of age have significantly higher levels of lipoprotein(a) in serum than men of the corresponding age was unexpected and must be studied more deeply, especially in the light of the negative effects of haemodialysis, i. e. the increase of serum lipoprotein(a) concentrations during dialysis, both when using chronic haemodialysis (23, 24) or continuous ambulatory peritoneal dialysis (25).

As there is no long-term treatment for increased lipoprotein(a) levels, perhaps with the exception of HELP apheresis, the only course of action in such individuals, especially children, is to minimise the other cardiovascular risk factors as far as is possible (26). Whether there is an influence of the genetically determined apolipoprotein(a) forms on the risk of cardiovascular disease has still to be confirm, although results on haemodialysis patients tend to support such a claim (27).

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